ON THE PRIMACY OF MOLECULAR PROCESSES IN DETERMINING RESPONSE RATES UNDER VARIABLE-RATIO AND VARIABLE-INTERVAL SCHEDULES

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This study focused on variables that may account for response-rate differences under variable-ratio (VR) and variable-interval (VI) schedules of reinforcement. Four rats were exposed to VR, VI, tandem VI differential-reinforcement-of-high-rate, regulated-probability-interval, and negative-feedback schedules of reinforcement that provided the same rate of reinforcement. Response rates were higher under the VR schedule than the VI schedule, and the rates on all other schedules approximated those under the VR schedule. The median reinforced interresponse time (IRT) under the VI schedule was longer than for the other schedules. Thus, differences in reinforced IRTs correlated with differences in response rate, an outcome suggestive of the molecular control of response rate. This conclusion was complemented by the additional finding that the differences in molar reinforcement-feedback functions had little discernible impact on responding.

Key words: variable-ratio schedule, variable-interval schedule, molecular accounts, molar accounts, lever press, rats

Under a variable-interval (VI) 6-min schedule of reinforcement, pigeons make a response every second or two (Catania & Reynolds, 1968) even though the rate of reinforcement this schedule provides is largely unchanged across a wide range of response rates. From an optimizing perspective, these response rates are surprisingly high. Yet they appear moderate when compared to variable-ratio (VR) schedules where response rates are often 50% higher even when they provide the same rate of reinforcement as a VI schedule (e.g., Baum, 1993; Catania, Matthews, Silverman, & Yohalem, 1977; Ferster & Skinner, 1957; Matthews, Shimoff, Catania, & Sagvolden, 1977; Peele, Casey, & Silberberg, 1984; Zuriff, 1970).

How are these two effects to be explained? In their account of the moderate rates supported by VI schedules, Silberberg, Warren-Boulton, and Asano (1988) referred to the impulsivity that may operate at the level of the interresponse time (IRT). As applied to Catania and Reynolds' (1968) results, their account suggests that pigeons make many short IRTs even though their prospects for reinforcement are low because short IRTs produce scheduled reinforcers sooner than do long IRTs. Shimp (1969) had shown previously that the relative frequency of an IRT approximates its relative harmonic length. Thus, animals are biased in their IRT emission, favoring short IRTs and the proximal reinforcers they deliver over longer IRTs that are distally reinforced even when the reinforcement likelihoods for both types of IRTs are the same.

On VI schedules, the probability of reinforcement is an increasing and bounded function of IRT duration. If response emission is controlled by this relation (*molecular control*), the simplest expectation is that response rates should be low. Nevertheless, moderate response rates obtain under these schedules, and short IRTs may predominate because animals' preference for reinforcement now over later can only be realized by short-IRT emission, as Silberberg et al. (1988) argued. By this argument, the moderate rates of responding seen under VI schedules are not a violation of the notion of molecular control but a reflection of it.

A similar argument can be advanced to explain the high rates of responding seen under VR schedules. On first consideration, it would seem that control would not occur at the molecular level because the probability of reinforcement under these schedules is un-

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changed with IRT duration (Morse, 1966). But based on the argument offered above, a more appropriate way to understand VR responding is that, unlike the case for VI responding, molecular control does not constrain animals' preferences for short-IRT emission through differential reinforcement of long IRTs. Unconstrained by these molecular factors, VR response rates can be high, populated almost entirely by IRTs of short duration as animals are more immediately reinforced for short IRTs.

Peele et al. (1984) showed that manipulations of reinforced IRTs can cause responding under VR schedules to look VI-like and responding under VI schedules to look VR-like. These demonstrations showcase the sensitivity of response emission to the differential reinforcement of IRTs, but some researchers have questioned whether molecular control alone accounts for VR–VI rate differences (Cole, 1994; Dawson & Dickinson, 1990). If, in fact, the VR–VI rate difference is not exclusively controlled by between-schedule differences in reinforced IRTs, then what else may be involved?

An alternative approach was offered by Baum (1981), who attributed the VR–VI response-rate difference to differences in the relation between the rates of response and reinforcement that each type of schedule engenders (*molar control*). He noted that marginal increases in response rate are more productive of higher rates of reinforcement under VR schedules than VI schedules. Therefore, if response rate is controlled by the degree of correlation between response rate and reinforcer rate, then animals should respond more rapidly under VR schedules than VI schedules—the result that typically obtains.

Several synthetic schedules have been created to evaluate whether, and in what ways, response rate is sensitive to molar control. In some of these schedules, the rate of reinforcement was positively correlated with response rate (Cole, 1999; McDowell & Wixted, 1986; Reed, Hildebrandt, DeJongh, & Soh, 2003; Reed, Soh, Hildebrandt, DeJongh, & Shek, 2000). In others there was an inverse relation between these variables (Ettinger, Reid, & Staddon, 1987; Jacobs & Hackenberg, 2000; Reed & Schachtman, 1991; Vaughan, 1987; Vaughan & Miller, 1984). In still others, reinforcer rate was largely independent of response rate (Dawson & Dickinson, 1990; Kuch & Platt, 1976). The conclusions of these studies are mixed. Some argue that molar response-rate/reinforcer-rate correlations affect response rate (e.g., Dawson & Dickinson, 1990), but others largely deny this claim (e.g., Cole, 1999; Ettinger et al., 1987).

What makes it difficult to choose between these two views, at least as they relate to the VR–VI response-rate difference, is that often these studies addressed questions other than the origins of the difference. Given their alternate purposes (e.g., evaluating optimality accounts of behavior), some of the studies did not even include VR and VI schedules. So, although they may have addressed whether molar feedback functions, such as those involved in response-rate/reinforcer-rate correlations, affect responding, their claims may be restricted to the specific schedules used in their study. A further complication lay in the possibility that the structure of the feedback function may affect the sensitivity of response rate to manipulations of response-rate/reinforcer-rate correlations. For example, when the feedback function is negative (increases in response rate produce decreases in reinforcement rate), molar control uniformly fails (e.g., Ettinger et al., 1987). Indeed, it is only when reinforcer rate is independent of response rate or is positively correlated with it that evidence of molar control has appeared (see, e.g., Dawson & Dickinson, 1990; McDowell & Wixted, 1986; Reed, 2006; Soto, McDowell, & Dallery, 2006).

The large number of different molecular and molar manipulations used to determine the source of the VR–VI response-rate difference complicates the development of a coherent summary of the literature. In addition, even when the same manipulation occurred across laboratories, methodological differences remain, ranging from the species used to the presence or absence of multiple schedules to exactly how a particular schedule was defined. In consequence, on those occasions where a data set is at variance with other findings, it is sometimes difficult to assign cause to effect.

Despite these difficulties, our reading of the literature is generally supportive of the conclusion reached by Peele et al. (1984) that the ratio–interval rate difference is primarily due

to between-schedule differences in IRT reinforcement. A major goal of the present work was to address those data sets that are problematic for this conclusion. First, Dawson and Dickinson (1990) found that their regulated-probability-interval (RPI) schedule, which had the molar feedback properties of an interval schedule but reinforced IRTs in a fashion akin to a VR, supported lower response rates than a random-ratio schedule. Surely, if molecular control through between-schedule differences in IRT reinforcement explains the ratio–interval rate difference, this outcome should not obtain. Second, Cole's (1994) finding that tandem VI differential-reinforcement-of-high-rates (DRH; tandem VI DRH) schedules did not support ratio-like response rates is incompatible not only with other data (Peele et al., 1984) but also with molecular control of the VR–VI rate difference. Finally, Reed et al. (2000, Experiment 4) found that rates under a tandem VI+ DRH schedule were lower than those under VR. Given that a VI+ schedule is defined to have the molar feedback function of a VR, and the DRH was defined to produce the molecular reinforcement likelihoods of a VR, this finding is actually incompatible with both a molar and a molecular account of response rates.

The present experiment addressed the three problematic outcomes described above through an examination of performances on two schedules, an RPI schedule and a tandem VI DRH schedule. The RPI schedule was a direct replication of that used by Dawson and Dickinson (1990). As regards the tandem VI DRH and tandem VI+ DRH schedules, only the former was evaluated. To anticipate our results, we will show that the tandem VI DRH schedule used by Cole (1994) supports response rates similar to those of a VR. Given that the tandem VI+ DRH schedule used by Reed et al. (2000) differs only in that it has a VR-like molar feedback function, it, too, would presumably support a response rate no lower than that seen in our tandem VI DRH data. For that reason, a test involving the tandem VI+ DRH schedule was not performed.

Although our emphasis was on the three schedule types noted above, the present study also reexamined the negative feedback (NF) schedule used by Reed and Schachtman (1991). In Experiment 3 of their report, they exposed rats to a tandem NF differential-reinforcement-of-low-rates (DRL) schedule in which the DRL criterion was equated to the reinforced IRT found in a prior VI condition. They found that the response rates obtained were similar to those seen under VI schedules. The results endorse the molecular control of response rate differences, but they do not address a complementary question: If IRT reinforcement approximated that seen under a VR schedule, would the response rates be VR-like even though the molar feedback function differentially reinforced low response rates? The present study addressed this question by examining response rates maintained by an NF schedule that shared with a standard VR schedule the molecular property of probability of reinforcement being independent of IRT duration. If response rates under this schedule matched those under a comparison VR schedule, then these data would support the view that response emission is under molecular control whether IRT reinforcement is similar to that seen under VI schedules, as in Reed and Schachtman's (1991) study, or similar to that seen under VR schedules. Alternatively, should response rates fall short of those maintained by the comparison VR schedule, then this result would identify selective insensitivity in molecular control.

METHOD

Subjects

Four experimentally naïve, male Wister albino rats served. Subjects were approximately 3 months old at the start of training, maintained at 80% of their free-feeding weights, and housed individually in a temperature-controlled room on a 12-hr light/12-hr dark cycle with free access to water.

Apparatus

An operant chamber (31 cm long by 20 cm wide by 25 cm high) was housed in a sound- and light-attenuating enclosure. Extraneous sounds were masked by a ventilating fan. A lever that required a force of approximately 0.3 N to operate was on the left side of one wall of the chamber, 4.7 cm above the grid floor and 4.5 cm from the left side wall. A 2.8-W, white light was located 2.8 cm above the lever. The center of a 3-cm diameter food cup, which

Table 1

Summary of conditions and results for each rat.

			Rats				
	Condition	Schedule	T1	T2	T3	T4	Mean
Number of sessions	1	VR	38	30	32	37	34.25
	2	RPI	23	22	20	20	21.25
	3	NF	20	23	21	21	21.25
	4	Tand VI DRH	21	22	21	21	21.25
	5	VR	20	21	21	21	20.75
	6	VI	37	37	37	33	36.00
	7	NF	36	24	24	31	28.75
	8	VR	20	22	20	20	20.50
Responses/min	1	VR	80.05	50.52	51.59	54.73	59.22
	2	RPI	75.28	53.58	52.69	67.69	62.31
	3	NF	76.38	48.13	49.32	69.10	60.73
	4	Tand VI DRH	75.29	67.52	47.93	56.08	61.71
	5	VR	81.47	68.69	51.33	62.14	65.91
	6	VI	62.14	57.14	42.60	47.97	52.46
	7	NF	64.37	71.41	53.25	57.71	61.69
	8	VR	65.76	72.52	47.87	47.72	58.47
Reinforcers/min	1	VR	2.53	1.69	1.70	1.76	1.92
	2	RPI	2.54	1.51	1.58	1.69	1.83
	3	NF	2.62	1.65	1.66	1.03	1.74
	4	Tand VI DRH	2.38	1.60	1.54	1.66	1.79
	5	VR	2.80	2.36	1.67	2.11	2.24
	6	VI	2.58	2.27	1.61	2.04	2.12
	7	NF	3.10	2.00	1.47	2.03	2.15
	8	VR	2.20	2.36	1.62	1.56	1.93
Median reinforced IRT length (s)	1	VR	0.51	0.87	0.68	0.92	0.74
	2	RPI	0.45	0.79	0.83	0.65	0.68
	3	NF	0.37	0.91	0.87	0.67	0.70
	4	Tand VI DRH	0.31	0.50	0.48	0.61	0.47
	5	VR	0.41	0.58	0.82	0.78	0.65
	6	VI	1.41	1.42	1.83	1.39	1.51
	7	NF	0.46	0.56	0.91	0.85	0.69
	8	VR	0.44	0.46	0.83	1.00	0.68
Median length of IRT prior to the one reinforced (s)	1	VR	0.44	0.88	0.81	0.92	0.76
	2	RPI	0.40	0.91	0.92	0.72	0.74
	3	NF	0.39	0.98	0.80	0.83	0.75
	4	Tand VI DRH	0.88	1.20	1.11	1.09	1.07
	5	VR	0.39	0.60	0.77	0.82	0.64
	6	VI	0.58	0.66	0.98	0.92	0.78
	7	NF	0.51	0.56	0.72	0.78	0.64
	8	VR	0.74	0.41	0.89	0.95	0.75

presented 45-mg food pellets, was positioned 5.5 cm to the right of the center of the response lever. A 2.8-W, white houselight was located in the center of the ceiling of the chamber. A computer recorded all responses in 0.1-s intervals and controlled all experimental events.

Procedure

The rats were trained to lever press by successive approximation and then exposed to a pretraining phase consisting of two sessions of exposure to each of the following schedules: continuous reinforcement, VR 5, VR 10, VR 15, and VR 20 in that order. The 15 ratios comprising each of the VR schedules were derived using the Fleshler and Hoffman (1962) progression and were randomly sampled without replacement. Reinforcers consisted of a single 45-mg food pellet. After a pellet was delivered to the food cup, the lever light was extinguished for 2 s. Each pretraining session ended after 40 reinforcers had been delivered.

The rats were then introduced to the eight experimental conditions defining this study (see Table 1). Four of the conditions presented VR 30 (Conditions 1, 5, and 8) or VI

(Condition 6) schedules. Conditions 2 and 4, respectively, presented RPI and tandem VI DRH schedules, and Conditions 3 and 7 presented NF schedules.

RPI schedule. The molar properties of an RPI schedule are illustrated in Figure 1. This schedule was defined by first exposing a rat to a VR schedule (top left panel) and calculating the reinforcer rate that that schedule provided at the response rate the animal produced (top right panel). As shown by the dashed line in the bottom left panel, the reinforcer rate the RPI schedule provides is largely independent of response rate.

Under this schedule, the nth response was reinforced according to the following equation:

$$P_n(rft) = R_{VR}t/m, \quad (1)$$

where $P_n(rft)$ is the probability of reinforcement for the nth response, R_{VR} is the average reinforcement rate during the last five sessions of Condition 1 (VR schedule) for each rat, t is the total duration from the $(n - 1 - m)$ IRT to the $(n - 1)$ IRT, and m is the IRT memory size. IRT memory size was set at 50, the same value used by Dawson and Dickinson (1990). Therefore, t/m equaled the average duration of the last 50 IRTs with a lag of one. The definition of t meant that $P_n(rft)$ was independent of the IRT of the nth response and should result in RPI reinforcer rates that were the same as R_{VR} (see the Appendix for a fuller explanation).

NF schedule. This schedule also is illustrated in Figure 1. As shown by the dashed line in the bottom right panel, increases in response rate result in decreases in reinforcer rate. These schedules use the average duration of the most recent IRTs with a lag of one to determine reinforcer rate. The probability of reinforcement was defined as:

$$P_n(rft) = (R_{VR} + B_{VR}/30)t/m - 1/30, \quad (2)$$

where B_{VR} is the average response rate from the last five sessions of Condition 1 for each rat, and the other terms (t, m, and R_{VR}) have the same meanings as in Equation 1. The last term in the equation defines the slope of the schedule's feedback function. Reinforcement rates under the NF schedule were expected to be the same as R_{VR} when response rates were the same as B_{VR}. Equation 2 also was used in Condition 7, but in that case the values of R_{VR} and B_{VR} were based on the data from the last five sessions in Condition 5 (see the Appendix).

Tandem VI DRH and VI schedules. The VI values that comprised the tandem VI DRH and VI schedules (Conditions 4 and 6) were determined by a within-subject yoked-control procedure (Peele et al., 1984). For each rat, all interreinforcement intervals (IRIs) from the last five sessions of Condition 1 were recorded and were then used to define the successive IRIs in the VI component of the tandem VI DRH schedule. The same procedure was used to define the intervals of the VI schedule in Condition 6, but those values were based on the data from the VR schedule in Condition 5.

The DRH values in the DRH component of the tandem schedule were defined for each rat in the following way: First, the duration of each reinforced IRT during the last five sessions of Condition 1 was recorded, and the median and interquartile values from the IRT distribution were used to define the duration of four quartile-based IRT classes: 0–0.25, 0.25–0.5, 0.5–0.75, and 0.75–1.0. These IRT classes then defined the criterion for reinforcement during the DRH component. For example, if the consecutive values of the reinforced IRTs under the VR schedule were 0.1, 0.6, 0.3, 1.2, and 0.1 s, then DRH values of 0.25, 0.75, 0.5, infinity (that is, any IRT was reinforced), and 0.25 s, in that order, would be the longest IRTs that could produce reinforcement (also see Cole, 1994; Peele et al., 1984).

Each condition lasted until the following criteria were met: (a) at least 20 sessions had elapsed; (b) response rates in each of the last five sessions were within ±10% of the five-session average; and (c) no trend was apparent by visual inspection. Daily sessions were conducted five or six times per week.

In all other regards, the eight conditions of the main experimental phase were the same as in pretraining (e.g., sessions ended after 40 reinforcers had been obtained).

RESULTS

Table 1 summarizes the results of the present experiment. The values other than number of sessions are based on the last five sessions in each condition. Reinforcer rates under the RPI, first NF, and tandem VI DRH schedules (Conditions 2, 3, and 4) approxi-

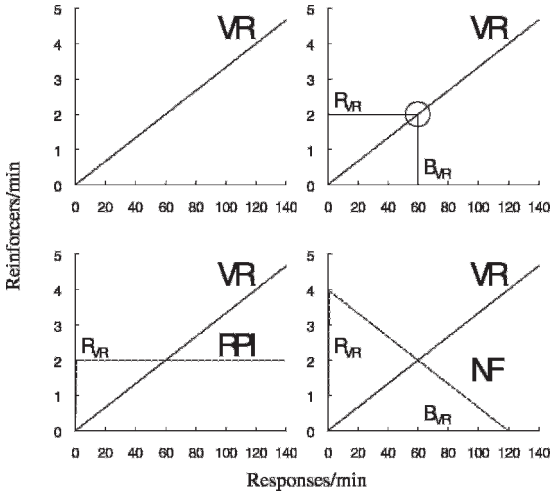

Fig. 1. Reinforcers per min as a function of responses per min presented as molar feedback functions. The top two panels present the feedback function for VR (left panel) and the determination of a specific reinforcer rate at a given response rate (right panel). The bottom panels present the feedback function for the RPI (left panel) and NF (right panel) schedules. The symbols R_{VR} and B_{VR} that appear represent values of the variables in Equations 1 and 2.

mated those during the first VR schedule (Condition 1). Similarly, the reinforcer rates were approximately equal under the second VR schedule (Condition 5) and the VI and second NF schedules (Conditions 6 and 7). To a first approximation, it appears that the RPI, NF, tandem VI DRH, and VI schedules produced a reinforcer rate roughly comparable to their target (first or second) VR schedule.

The near-equivalence in mean reinforcer rates was, with the exception of the VI schedule, largely matched by the mean response rates these schedules produced. In three of four cases, the mean response rate under the VI schedule was the lowest for any of the schedules.

As shown in Table 1, median reinforced IRTs under the VI schedule were longer than those under the other schedules, where the median reinforced IRTs were approximately the same as that for the target VR schedules.

Finally, the median durations of the IRT just prior to the reinforced IRT also are presented in Table 1. The median just-prior IRT under the tandem VI DRH was substantially longer than that for the other schedules even though the mean response rate under this schedule was roughly the same as under the other schedules, excepting the VI.

Figure 2 presents the relation between reinforcer rate and response rate in each of the eight experimental conditions. The condition number appears in parentheses. Performance in all sessions of each condition is represented in the figure. Lines of best fit on a least-squares criterion also are shown for each subject. These best-fitting lines provide an index of the effectiveness of the molar feedback functions. Overall, the best-fitting lines were positive for the VR schedules, approximately flat for the RPI, VI, and tandem VI DRH schedules, and negative for the NF schedules. Indeed, Table 2 shows there was a close correspondence between the idealized and actual slopes of the feedback functions presented in the figure. To make the method of calculating the idealized slope explicit, consider the case of VR 30. Since it requires 30 responses, on average, to produce a reinforcer, mapping the feedback function between reinforcement and behavior is defined as one reinforcer per 30 responses or 0.033. The same approach was used to define the idealized slopes for the other schedules.

Figure 3 presents the mean response-rate of the last five sessions for each rat across the five types of schedules presented in this experiment. When multiple points appear in a panel, it signifies that a particular condition was redetermined. Note the differences in the y-axis for different subjects. Except for Rat T2, response rates under the VI schedule were almost always lowest, rates under the VR schedule were higher, and rates to the other three schedules usually were closer to those under the VR than the VI. Moreover, even for Rat T2, only the response rate under the RPI schedule clearly violated the general conclusion that the synthetic schedules used in this study tended to produce response rates that were more ratio- than interval-like. Since, in all these cases, the operation of the molecular factor of differential IRT reinforcement should favor higher rates, it seems sensible to conclude that the present study's findings support a molecular account of the ratio-interval rate difference.

DISCUSSION

The IRTs reinforced under the VI schedule were approximately twice as long as those

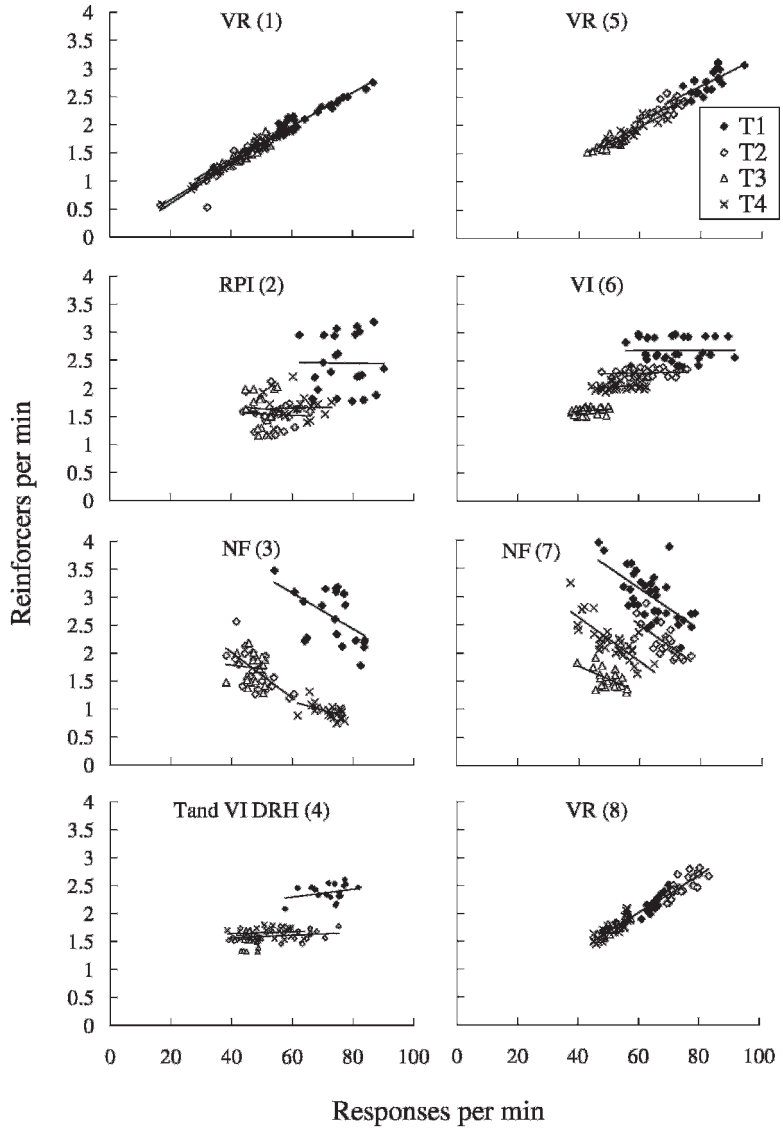

Fig. 2. Reinforcers per min as a function of responses per min in the eight experimental conditions. See text for additional details.

obtained in all the other schedules evaluated in this experiment. The fact that the VI schedule also produced a mean response rate that was lower than the other schedules (see Table 1 and Figure 3) is evidence of the influence of molecular contingencies in controlling response rate in this study and endorses the claim that the ratio–interval rate difference is attributable to this factor.

The selection of the schedules used in the present study was largely based on those data sets in the literature that were incompatible with the claim that the molecular factor of differential IRT reinforcement accounts for the ratio–interval rate difference. Our replication of this work makes it possible to unify all these schedule effects as evincing the molecular control of response rate. Of course, we cannot explain why the original data were discrepant from a molecular-control account, but the finding that they were not replicated may obviate the discrepancy.

Table 2

Idealized and actual slopes of the feedback functions in each condition.

Condition	Schedule	Ideal slope	Actual slope			
			T1	T2	T3	T4
1	VR	0.033	0.030	0.036	0.031	0.032
2	RPI	0.000	−0.001	−0.002	−0.005	0.001
3	NF	−0.033	−0.033	−0.041	−0.007	−0.015
4	Tand VI DRH	0.000	0.007	0.001	−0.002	0.001
5	VR	0.033	0.027	0.023	0.018	0.027
6	VI	0.000	−0.002	0.001	0.005	0.003
7	NF	−0.033	−0.020	−0.038	−0.023	−0.039
8	VR	0.033	0.032	0.035	0.034	0.036

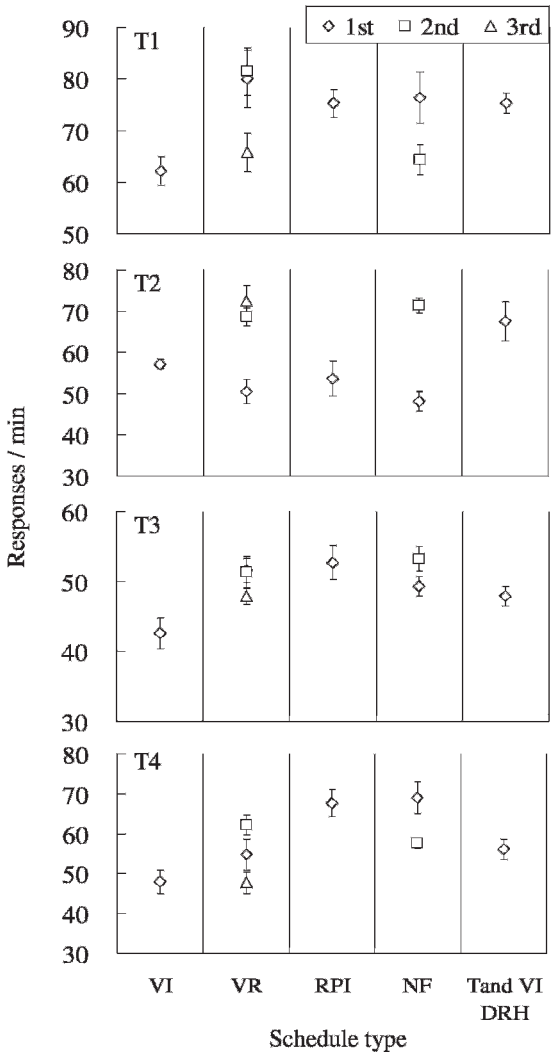

Fig. 3. Responses per min for the different schedule types for each rat. Multiple points appear where the schedule was reintroduced. The bars show the standard deviation from the last five sessions in each condition.

The present results provide further support for the view that the molecular relationship between IRT duration and the probability of reinforcement is the primary determinant of the difference in response rate between VR and VI schedules of reinforcement. Nevertheless, one feature of our findings suggests a possible problem with this conclusion. As shown in Table 1, the median duration of the IRT just prior to the reinforced IRT was longest under the tandem VI DRH schedule compared to the other schedules and more than twice as long as the median duration of the reinforced IRT under the same tandem VI DRH schedule. This result raises the possibility that the contingencies that define this schedule generated a particular pattern of responding, namely, bouts of high response rate interspersed with longer IRTs. This possibility may give pause to those who manipulate reinforced IRT distributions in order to test the molecular control of response rates. Our data suggest that overall response rate may result from two or more different response patterns. Resolving this question will require further research (see, e.g., Shull, Gaynor, & Grimes, 2001).

The analysis we have offered here views molar and molecular accounts as categorical. In one sense, this characterization is correct as long as the comparison is between reinforcement of a single IRT at one extreme and response-rate/reinforcer- rate correlations at the other extreme. However, as the reader is no doubt aware, sometimes schedules are not defined in ways compatible with categories. If a DRH or DRL schedule is not based not on a single response but on multiple responses (e.g., defining the DRH contingency as the emission of four responses in 5 s, or the DRL contingency as fewer than two responses in

5 s), the schedules so defined begin to assume a more molar definition based on response rate. Similarly, molar schedules can become molecular-like. For example, Soto et al. (2006) examined a VI schedule with what they called a ''quadratic feedback function'' and demonstrated response-rate control under this molar schedule. Although their results demonstrated molar control of responding—a result distinct from the conclusions of this article—it should be stressed that each response had such an abrupt effect on reinforcement likelihoods that it could be credibly claimed that their ostensibly molar test actually may have been a blend of molar and molecular contingencies. In our view, tests of molar versus molecular control are better served when the schedules being compared reside at the endpoints of the molar-to-molecular contingency continuum.

REFERENCES

Baum, W. M. (1981). Optimization and the matching law as accounts of instrumental behavior. *Journal of the Experimental Analysis of Behavior, 36*, 387–403.

Baum, W. M. (1993). Performances on ratio and interval schedules of reinforcement: Data and theory. *Journal of the Experimental Analysis of Behavior, 59*, 245–264.

Catania, A. C., Matthews, T. J., Silverman, P. J., & Yohalem, R. (1977). Yoked variable-ratio and variable-interval responding in pigeons. *Journal of the Experimental Analysis of Behavior, 28*, 155–161.

Catania, A. C., & Reynolds, G. S. (1968). A quantitative analysis of the responding maintained by interval schedules of reinforcement. *Journal of the Experimental Analysis of Behavior, 11*, 327–383.

Cole, M. R. (1994). Response-rate differences in variable-interval and variable-ratio schedules: An old problem revisited. *Journal of the Experimental Analysis of Behavior, 61*, 441–451.

Cole, M. R. (1999). Molar and molecular control in variable-interval and variable-ratio schedules. *Journal of the Experimental Analysis of Behavior, 71*, 319–328.

Dawson, G. R., & Dickinson, A. (1990). Performance on ratio and interval schedules with matched reinforcement rates. *The Quarterly Journal of Experimental Psychology, 42B*, 225–239.

Ettinger, R. H., Reid, A. K., & Staddon, J. E. R. (1987). Sensitivity to molar feedback functions: A test of molar optimality theory. *Journal of Experimental Psychology: Animal Behavior Processes, 13*, 366–375.

Ferster, C. B., & Skinner, B. F. (1957). *Schedules of reinforcement*. New York: Appleton-Century-Crofts.

Fleshler, M., & Hoffman, H. S. (1962). A progression for generating variable-interval schedules. *Journal of the Experimental Analysis of Behavior, 5*, 529–530.

Jacobs, E. A., & Hackenberg, T. D. (2000). Human performance on negative slope schedules of points exchangeable for money: A failure of molar maximization. *Journal of the Experimental Analysis of Behavior, 73*, 241–260.

Kuch, D. O., & Platt, J. R. (1976). Reinforcement rate and interresponse time differentiation. *Journal of the Experimental Analysis of Behavior, 26*, 471–486.

Matthews, B. A., Shimoff, E., Catania, A. C., & Sagvolden, T. (1977). Uninstructed human responding: Sensitivity to ratio and interval contingencies. *Journal of the Experimental Analysis of Behavior, 27*, 453–467.

McDowell, J. J., & Wixted, J. T. (1986). Variable-ratio schedules as variable-interval schedules with linear feedback loops. *Journal of the Experimental Analysis of Behavior, 46*, 315–329.

Morse, W. H. (1966). Intermittent reinforcement. In W. K. Honig (Ed.), *Operant behavior: Areas of research and application* (pp. 52–109). New York: Appleton-Century-Crofts.

Peele, D. B., Casey, J., & Silberberg, A. (1984). Primacy of interresponse-time reinforcement in accounting for rate differences under variable-ratio and variable-interval schedules. *Journal of Experimental Psychology: Animal Behavior Processes, 10*, 149–167.

Reed, P. (2006). Effect of required response force on rats' performance on a VI+ schedule of reinforcement. *Learning & Behavior, 34*, 379–386.

Reed, P., Hildebrandt, T., DeJongh, J., & Soh, M. (2003). Rats' performance on variable-interval schedules with a linear feedback loop between response rate and reinforcement rate. *Journal of the Experimental Analysis of Behavior, 79*, 157–173.

Reed, P., & Schachtman, T. R. (1991). Instrumental performance on negative schedules. *The Quarterly Journal of Experimental Psychology, 43B*, 177–197.

Reed, P., Soh, M., Hildebrandt, T., DeJongh, J., & Shek, W. Y. (2000). Free-operant performance on variable interval schedules with a linear feedback loop: No evidence for molar sensitivities in rats. *Journal of Experimental Psychology: Animal Behavior Processes, 26*, 416–427.

Shimp, C. P. (1969). The concurrent reinforcement of two interresponse times: The relative frequency of an interresponse time equals its relative harmonic length. *Journal of the Experimental Analysis of Behavior, 12*, 403–411.

Shull, R. L., Gaynor, S. T., & Grimes, J. A. (2001). Response rate viewed as engagement bouts: Effects of relative reinforcement and schedule type. *Journal of the Experimental Analysis of Behavior, 75*, 247–274.

Silberberg, A., Warren-Boulton, F. R., & Asano, T. (1988). Maximizing present value: A model to explain why moderate response rates obtain on variable-interval schedules. *Journal of the Experimental Analysis of Behavior, 49*, 331–338.

Soto, P. L., McDowell, J. J., & Dallery, J. (2006). Feedback functions, optimization, and relation of response rate to reinforcer rate. *Journal of the Experimental Analysis of Behavior, 85*, 57–71.

Vaughan, W., Jr. (1987). Dissociation of value and response strength. *Journal of the Experimental Analysis of Behavior, 48*, 367–381.

Vaughan, W., Jr, & Miller, H. L., Jr. (1984). Optimization versus response-strength accounts of behavior. *Journal of the Experimental Analysis of Behavior, 42*, 337–348.

Zuriff, G. E. (1970). A comparison of variable-ratio and variable-interval schedules of reinforcement. *Journal of the Experimental Analysis of Behavior, 13*, 369–374.

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Appendix

This appendix expands the definition of two of the schedules described in the Method section.

The RPI schedule. The RPI reinforcer rate defined by Equation 1 will be the same as R_{VR}. The reinforcer rate is the product of the reinforcer probability and the response rate—that is,

$$R = P(rft)B. \quad \text{(A1)}$$

Substituting Equation 1 yields

$$R = R_{VR}(t/m)B, \quad \text{(A2)}$$

where t / m equals the average duration of the last 50 IRTs with a lag of one. This value is expected to equal $1/B$. If this obtains, then

$$R = R_{VR}(1/B)B = R_{VR}. \quad \text{(A3)}$$

The NF schedule. The negative feedback function was created by subtracting the reinforcer probability under a random-ratio schedule from that under an RPI schedule. That is:

$$P(rft) = R(t/m) - 1/30, \quad \text{(A4)}$$

where R is the y-intercept. The goal was to ensure that reinforcer rates under the NF schedules were the same as R_{VR} when response rates were the same as B_{VR}. This was achieved as follows:

$$R_{VR} = R - B_{VR}/30. \quad \text{(A5)}$$

Rearranging this equation for R results in

$$R = R_{VR} + B_{VR}/30. \quad \text{(A6)}$$

Substituting this equation into Equation A.4 yields Equation 2.